Amendments to the Drawings:

Attached hereto, on four Replacement Sheets, are revised drawing Figures 2A, 2B, 3 and 5, which have been amended for brightness and clarity. No new matter has been introduced.

REMARKS

Claims 5-8 and 21-26 are pending in this application. Claims 1-4, 9-20, and 27 were canceled without prejudice or disclaimer of the subject matter therein in the previous response. By this amendment, several claims have been amended, new claims 28-30 have been added. No new matter has been introduced. Claims 5-8 and 21-26 remain pending.

Election/Restrictions

Applicants note with appreciation the indication that the election of Group I was acknowledged. The Action indicates that Applicants' February 22, 2006 response was treated as one without traverse since it did not distinctly and specifically point out the error in the restriction requirement. Applicants respectfully assert that the February 22, 2006 Response was a supplemental response and that the original response, filed December 29, 2006 contained Applicants reasoning with regard to traverse, thus, preserving the right to appeal the restriction requirement. The point is made for clarity of the record, and has no bearing here, since Applicant canceled all non-elected claims in the February 22, 2006 response.

Information Disclosure Statement

We note with appreciation the indication that the February 19, 2004, May 7, 2004 and December 20, 2005 IDSs have all been considered by the Examiner. The Examiner has also indicated, by attaching initialed copies, consideration of the February 22, 2006 IDS. We note, however, that an initialed copy of the December 20, 2005 IDS was not attached to the current action, nor does one appear in PAIR. A copy of the December 20, 2005 IDS is readily available in PAIR. *Applicants respectfully request that the Examiner provide an initialed copy of the December 20, 2005 IDS with the next action.*

Request for Corrected Priority

Applicants respectfully request correction of the priority listed for this application. The filing receipt indicates priority to U.S. Provisional Application Serial No. 60/156,333 filed on September 28, 1999. Applicants respectfully submit that through inadvertent typographical error, the application number is incorrect. The correct serial number is 60/156,633 which was filed on September 29, 1999. Applicants submit herewith an updated Application Data Sheet,

reflecting the corrected information. Applicants respectfully request that the priority information be updated accordingly.

Drawings

Applicants submit herewith revised version of Figs. 2A, 2B, 3, and 5. No new matter has been added. The new figures have been improved for better visibility. Applicants respectfully submit that the new figures are now suitable for publication.

New Claims

New claims 28 to 30 have been added, without introducing new matter. New claims 28 and 29 parallel amended claims 4 and 6, but further claim the use of an endogenous version of the polypeptide of SEQ ID NO:20 which is encoded by a polynucleotide that hybridizes under stringent conditions to the complement of SEQ ID NO:19, wherein said stringent conditions comprise a wash at 65°C in 0.1xSSC. The stringent conditions are disclosed with reference to hRUP4 on page 28 of the original specification. Those of skill in the art will readily recognize what is meant by stringent conditions and that endogenous variants of SEQ ID NO:20 are encoded by polynucleotides which hybridize to the complement of SEQ ID NO: 19. Support for new claim 30 can be found generally throughout the specification and specifically at page 15, lines 9-12 of the original specification. Applicants respectfully submit that no new matter has been introduced by new claims 28 to 30.

Claim Rejections – 35 U.S.C. § 101

Claims 1-4, 9-20, and 27 stand rejected under 35 U.S.C. § 101 for allegedly lacking a well-established or asserted utility. As noted above, Applicants' last response canceled claims 1-4, 9-20 and 27. As such, the rejection, as written, is moot. Applicants respectfully request withdrawal of the rejection.

Nevertheless, since the rejection raises questions of utility which could be construed to affect the subject matter related to SEQ ID NO: 20 and hARE-2 receptor, generally, Applicants respectfully assert that there is either a well-established utility or an asserted specific, substantial, and credible utility.

The Utility

Applicants respectfully submit that those of skill in the art would readily recognize the utility of using the GPCR, hARE-2, in the treatment or identification of compounds for treatment of motor impairment disorders associated with the substantia nigra, such as Parkinson's disease.

The Utility is Specific

Applicants respectfully submit that the utility is specific. Each of the claims is directed specifically to hARE-2 and not to GPCRs generally. Despite the characterization in the Action, the disorders to be treated are also specific to diseases or disorders related to the substantia nigra, such as motor impairment disorders, including Parkinson's disease. The specification indicates that the localized expression data is used to determine where the receptor is expressed, and accordingly is associated with a functionality. In this case, expression was found in the substantia nigra--an area of the brain known to be correlated to motor function and motor impairment disorders. This knowledge coupled with the knowledge that modulation of hARE-2 (e.g., by a ligand identified through a screening assay that employs hARE-2) can lead to a modulation of cAMP or IP3 selectively in the substantia nigra, would lead those of ordinary skill in the art to recognize the identified ligand can be used specifically to treat a disease or disorder of the substantia nigra such as Parkinson's disease. Thus, the utility is specific as contemplated by 35 U.S.C. § 101.

The Utility is Substantial

The specification does not blindly recite the use of hARE-2 for treating an unknown disease or disorder, rather, the specification is clear that hARE-2 can be used to treat diseases and disorders associated with the substantia nigra, such as motor impairment diseases and disorders. The treatment of motor impairment disorders and, especially Parkinson's disease, is a real world use.

Several noteworthy celebrities have Parkinson's disease and have been outspoken in their search for a cure. In particular, in recent years, actor Michael J. Fox, who suffers from Parkinson's, has spoken before Congress, and other audiences, to raise awareness for the disease and the search for treatment and a cure. Such efforts are a clear demonstration of a cause having real world applications Many people suffer from this disabling disorder and would benefit from a treatment for the disease or even a treatment preventing or limiting further exacerbation of the disease

The utility is substantial because the use of hARE-2 in an assay to identify possible ligands for treating a disease or disorder of the substantia nigra such as Parkinson's disease is a "real world" use. In this regard, Applicants note that the Revised Interim Utility Guidelines Training Material (herein after "Training Material") states that "an assay method for identifying

compounds that themselves have a 'substantial utility' define a 'real world' context of use." See page 6 of the Training Material. In the present case, the ligands that can be identified in an assay employing hARE-2 have substantial utility themselves because, as disclosed by Applicants, these ligands can be administered to treat a disease or disorder of the substantia nigra, such as Parkinson's disease. Thus, the use of hARE-2 in an assay to identify ligands thereof for treating disorders of the substantia nigra also would have a "real world" use.

Thus, Applicants have disclosed a substantial, real world use as contemplated by 35 U.S.C. § 101.

The Utility is Credible

As mentioned above, it is the Office's burden to provide evidence tending to show that the utility is not credible. The Action offers no factual evidence to contravene Applicants' assertion of utility. Absent any factual evidence from the Office that would lead one to question the credibility of the utility, the Office must recognize the asserted utility. Applicants respectfully submit that all the requirements of 35 U.S.C. § 101 have been satisfied.

Those of skill in the art would have had no reason to question the use of the GPCR, hARE-2, which is expressed in the substantia nigra, to screen for ligands that could have been administered to a patient to treat a disease or disorder of the substantia nigra, such as Parkinson's disease.

The MPEP dictates that once the Applicants have provided a reason for why the claimed invention is useful, the Office personnel may maintain a rejection for alleged lack of utility *only* if the Office Action establishes that one of ordinary skill would find that the asserted utility is not credible. For example, the MPEP §2107.02 II. B. states that:

> If the applicant subsequently indicates why the invention is useful, Office personnel should review that assertion according to the standards articulated below for review of the credibility of an asserted utility.

The "standard articulated below for review of the credibility of an asserted utility " is found at MPEP §2107.02 III. B, which states:

> Where an applicant has specifically asserted that an invention has a particular utility, that assertion cannot simply be dismissed by Office personnel as being "wrong," even when there may be reason to believe that the assertion is not entirely accurate. Rather, Office personnel must determine if the assertion of utility is credible (i.e., whether the assertion of utility is believable to a person of ordinary skill in the art based on the totality of evidence and reasoning provided). An assertion is credible unless (A) the logic underlying the assertion is seriously flawed, or (B) the facts upon which the assertion is based are inconsistent with the logic underlying the assertion. Credibility as used in this context refers to the reliability of the statement based on the logic and facts that are offered by the applicant to support the assertion of utility. (Emphasis in original.)

The use of hARE-2 to screen for ligands of hARE-2, wherein such ligands can be administered to treat a disease or disorder of the substantia nigra, such as Parkinson's disease, would have been recognized by a person of ordinary skill in the art at least as early as the time of filing, upon reading Applicants' specification and claims. There is no evidence of record that would contradict the logic underlying this assertion or that would indicate that the facts upon which it is based, as set forth herein, are inconsistent with the logic underlying the assertion.

Thus, Applicants respectfully assert that those of skill in the art would have immediately recognized the utility of the invention and that 35 U.S.C. § 101 is satisfied.

Although Applicants believe that the question of utility is addressed above, Applicants continue, addressing the Action's specific comments.

The Action, alleges on page 4, that "the specification fails to disclose the ligand of the putative GPCR, fails to provide any sufficient information or evidence on the biological functions or activities of the hARE-2 polypeptide of SEQ ID NO: 20, and fails to disclose a patentable utility for the claimed invention." The Action continues, alleging lack of a well-established utility. The focus of the discussion is on the sequence homology of SEQ ID NO: 20 (hARE-2) with GPR27. The Action seems to question whether hARE-2 is a GPCR, but

ultimately indicates even if it were, there is still no biological function associated with hARE-2 and thus there is no well-established utility.

Applicants respectfully assert that those of skill in the art would readily recognize that hARE-2 is a GPCR as indicated by Applicants. Those of skill in the art at the time of filing would not need to rely solely on the sequence homology of hARE-2 with GPR27 to appreciate that hARE-2 is a GPCR. Applicants respectfully point out that Figure 1 of priority provisional application US 60/136,436 provides an alignment of hARE-2 with GPR27, where it was shown that the homology between hARE-2 and GPR27 is observed over the entire length of hARE-2. Applicants also respectfully point out that a person of skill in the art at least as early as the priority date of the application would readily have appreciated that hARE-2 is characterized by the 7 hydrophobic domains characteristic and highly conserved in GPCRs as well as by additional conserved features of GPCRs.

It was well known to the skilled artisan that GPCRs are characterized by seven transmembrane (membrane spanning) domains, designated TM-1 to TM-7 (*see, e.g.,* page 2, line 21 through page 3, line11 of the application as filed; and page 2, left column, lines 13-27, Figure 2, and Figure 3 of Probst et al. (1992) DNA Cell Biol., 11:1-20). It was well known to the skilled artisan that TM-6 is characterized by a tryptophan residue and a proline residue conserved in many GPCRs (*see, e.g.,* page 10, lines 7-10 of the application as filed; and page 2, left column, lines 32-40, to right column, lines 1-5, Figure 2, and Figure 3 of Probst et al.) It was well known to the skilled artisan that GPCRs are characterized by a highly conserved arginine residue at the intracellular end of TM3, typically as part of a perfect or imperfect "DRY" motif (*see, e.g.,* page 12, left column, lines 30-34, Figure 2, and Figure 3 of Probst et al.)

Methods of predicting the location of the transmembrane domains of a GPCR based on the sequence were available at least as early as the priority date of the application. One such method is TMHMM (transmembrane hidden Markov model), which is described in: Sonnhammer et al. (1998) In J Glasgow et al., eds, Proc Sixth Int. Conf. on Intelligent Systems for Molecular Biology, 175-182, enclosed. In order to demonstrate to the Examiner that known methods could have been applied by those skilled in the art to identify TM-1 to TM-7, the conserved tryptophan and proline residues in TM-6, and the highly conserved arginine residue at

the intracellular end of TM3, Applicants have applied the TMHMM method to SEQ ID NO:20 (hARE-2 amino acid sequence). The result is shown in **Annex 1**.

Annex 1 shows that hARE-2 is predicted to have seven transmembrane regions, as expected for a GPCR. **Annex 1** shows that TM-6 is predicted to correspond to amino acids 289-311 of SEQ ID NO:20. This predicted amino acid sequence for TM-6, shown in **Annex 2**, contains a tryptophan residue (amino acid position 299) and a proline residue (amino acid position 301), consistent with the tryptophan and proline residues which are conserved in TM-6 of many GPCRs. The highly conserved arginine residue adjacent to TM3 in the second can be found at amino acid position 121. It follows that it would be readily apparent to a person skilled in the art at least as early as the priority date that hARE-2 is a GPCR. The Office has provided no countervailing evidence to dispute this fact.

The Action continues, indicating that the specification lacks a specific, substantial, and credible utility, because the disclosure "fails to identify the ligand and the biological functions of the hARE-2 polypeptide: Thus, the Office's utility rejection is focused on a) the lack of a ligand for hARE-2 and b) the lack of a biological function for hARE-2.

Applicants respectfully submit that the disclosure of a ligand is NOT a prerequisite to a finding of utility. Knowledge of a GPCR's natural ligand ***is simply not necessary*** for establishing the function for such a receptor. In fact, it is possible to know a receptor's function and develop and market pharmaceutical agents targeting it without any understanding of the natural ligand which activates the receptor. For instance, the agonists of the so-called niacin receptor have long been recognized and used to raise HDL levels in man, e.g. nicotinic acid and acipomox. The natural ligand for this receptor was still a mystery until at least 2004. (Karpe and Frayn Lancet. 2004 Jun 5; 363(9424):1892-4). Similarly, many opiates were identified and developed and the analgesic functionality of the mu-opiate receptor was appreciated long before the first endogenous agonists of that receptor were discovered in 1975 (Zadina et al. Ann N Y Acad Sci. 1999; 897:136-44). Applicants may very likely develop and market modulators of hARE-2 without ever knowing hARE-2's natural ligand. It remains unclear to Applicant how knowledge of a receptor's natural ligand is a prerequisite to a finding of utility when such knowledge is not necessary to convince the FDA of the function of a drug which modulates the receptor, and is not necessary to treat real live human beings. The fact that such knowledge is

not required by the FDA is highly indicative that real world uses exist even where the natural ligand is unknown. Applicants note, however, that this entire line of reasoning is moot, since, for the reasons set forth in this response, the present application satisfies the utility requirement by asserting a specific, substantial, and credible utility and/or because a well-established utility was known in the art at the time of filing the application.

Those of Skill in the Art Would Have Immediately Appreciated Why the Invention is Useful

At least as early as the time of filing, a person of ordinary skill in the art would have immediately appreciated why the invention is useful. Those of skill in the art, e.g. biochemists and molecular biologists, would have immediately appreciated that the claimed invention directed to the receptor hARE-2 could have been used, for example, in an assay to identify a ligand that would have been useful to treat a disease or disorder of the substantia nigra such as Parkinson's disease. Those of skill in the art would have immediately appreciated that the claimed invention directed to the receptor hARE-2 would have been useful because:

(a) Applicants have shown that hARE-2 is a GPCR that is selectively expressed in the substantia nigra. See, for example, Table C of the application as filed (p. 27);

(b) those of skill in the art were well aware of GPCR screening methods that could be used to screen compounds against hARE-2. Exemplary screening methods are provided, e.g., on pages 10-15 of the application as filed;

(c) it was known that modulating a GPCR, such as hARE-2, can modulate the level of cAMP or IP_3 (see, e.g., pages 11-13 of the application as filed), such that modulating hARE-2 can modulate the level of cAMP or IP_3 selectively in the substantia nigra;

---

[1] Identification in Table C of hARE-2 expression also in left cerebellum and right cerebellum indicates that the tissue panel used to show selective expression of hARE-2 in substantia nigra was that of the RNA Master Blot™ grid shown in Figure 1B and associated text.

(d) it was known that an elevation of intracellular IP_3 can lead to an elevation of intracellular Ca^{2+} [Berridge, Nature (1993) 361:315-325];

(e) it was known that the viability of neurons in the substantia nigra is sensitive to the level of intracellular cAMP [Hulley et al., European Journal of Neuroscience (1995) 7:2431-2440] and to the level of intracellular Ca^{2+} [Hirsch et al., J Neural Transm Suppl (1997) 50:79-88];

(f) it was known that the pathological process behind the motor disabilities of Parkinsonism is a progressive degeneration of dopaminergic neurons of the substantia nigra, that results in dopamine depletion in the striatum. Brain dopamine deficiency is sufficient to explain all of the major symptoms of Parkinson's disease [see, e.g., the final two sentences of the Abstract in Blaszczyk, Acta Neurobiol Exp (Wars.) 58:79-93].

(g) thus, one skilled in the art, upon reading Applicants' specification, would have appreciated that modulating neuron viability in the substantia nigra would have been useful for treating a disease or disorder relating to degeneration of neurons of the substantia nigra, for example, Parkinson's disease;

(h) thus, those of skill in the art would have recognized that and would have had no reason to doubt that hARE-2 could have been used in an assay to identify a ligand that would have been useful for modulating substantia nigra function to treat a disease or disorder of the substantia nigra, for example Parkinson's disease.

Thus, a person of ordinary skill in the art would have immediately appreciated that the claims directed to hARE-2 would have at least one well-established utility, because hARE-2 can be employed in screening assays to identify, for example, ligands useful for treating a disease or disorder of the substantia nigra such as Parkinson's disease.

Applicants further respectfully submit that it was well-established in the art at least as early as the date of filing of the subject application that a ligand to a polypeptide selectively expressed in substantia nigra could be used in methods of radioimaging to detect Parkinson's disease (see, e.g., Leenders et al., Arch Neurol (1990) 47:1290-1298); Fischman et al. Synapse (1998) 29:128-141).

Parkinson's disease is caused by a loss of neurons in the substantia nigra. See e.g. Goodman & Gilman's The Pharmacological Basis of Therapeutics, Ninth Edition (1996) McGraw-Hill (p. 504, lines 6-10, left column) and Blaszczyk (1998) Acta Neurobiol Exp (Wars) 58:79-83 (see Abstract, lines 12-14), and Montastrue et al. (1996) Drugs & Aging 9:169-184 (p. 170, line 47, left column to line 2, right column.)

For example, ligands to dopamine transporter (expressed by viable neurons in substantia nigra) have been shown to be useful for detecting Parkinson's disease in methods of radio imaging. Leenders et al. show this to be the case with the dopamine transporter ligand 6-L-(18F)-fluorodopa.. Fischman et al. show this to be the case for the dopamine transporter ligand 2β-carbomethoxy-3β-(4-fluorophenyl)-n-(iodoprop-1-en-3-yl) nortropane.

Applicants have disclosed hARE-2 to be such a polypeptide selectively expressed in substantia nigra. Agonists, partial agonists, and inverse agonists of hARE-2 are ligands of hARE-2 and, as such, would be expected to be useful in methods of radioimaging for detecting Parkinson's disease. Thus, one of skill in the art would have recognized a well-established utility for hARE-2.

Applicants reiterate, that as written, the rejection under 35 U.S.C. § 101 is moot since it relates to previously canceled claims 1-4, 9-20, and 27. Applicants respectfully request that the above comments be taken into consideration in reevaluating the utility issues in the case with respect to the pending claims. To the extent that a 101 rejection is presented relating to the pending claims, Applicants respectfully submit that such an action should be non-final.

Claim Rejections – 35 U.S.C. § 112, second paragraph

Claims 5-8 and 21-26 stand rejected under 35 U.S.C. § 112, second paragraph for allegedly failing to particularly point out and distinctly claim the subject matter which Applicants regard as the invention.

Specifically, the Action states claims 5, 7, 21, and 24 are indefinite for three reasons:

First, the action indicates "it is unclear whether the claim requires contacting said one or more compounds merely with membrane of a host cell or the receptor present in the membrane." Applicants respectfully assert that, as alluded to in the Action, those of skill in the art would recognize that the receptor is found either in the host cell or in the host cell membrane. Accordingly, the claim calls for contact with either the host cell or the host cell membrane, either of which comprises the receptor. See page 11, lines 9-10 of the original specification, which teaches the use of isolated membrane in screening assays. The claim has been amended, without narrowing the claim, for clarity.

Second, the Action states that it is unclear what "functionality" in the phrase "inhibit or stimulate functionality of said receptor" is intended. Those of skill in the art will readily recognize that agonists and antagonists inhibit or stimulate the receptor. Applicants have amended the claims to clarify that it is the receptor that is inhibited or stimulated. Applicants respectfully assert that this amendment is not narrowing and is made merely to clarify the claim.

Third, the Action alleges that the steps of the methods do not necessarily achieve the goal set forth in the claim preamble, stating "it is unclear how a candidate modulator is determined and correlated to the preamble."

Applicants have amended the claims to include the additional step of "(c) identifying the compound or compounds that inhibit or stimulate said receptor as modulators of said receptor." This identifying step clearly correlates the compounds found to inhibit or stimulate the receptor with the goal of the preamble to identify modulators of the receptor.

The action also notes that with regard to claims 5-8, the goal of identifying an antagonist cannot be met, "because the polypeptide of SEQ ID NO: 20 is an orphan and its ligand is unknown." Without wishing to concede the point, Applicants have amended the claims to refer to "agonists, partial agonists, or inverse agonists" rather than to "modulators," support for which can be found e.g. at page 15, lines 10-11 of the original specification. The screening methods described herein and others known in the art, do not require knowledge of the receptor's natural ligand.

With respect to claims 21-26, using constitutively active GPCR, the Action alleges that the methods do not appear to be able to achieve the goal of identifying an agonist. Applicants assert that use of the constitutively active GPCR facilitates the identification of agonists (as well as partial agonists and inverse agonists). The Office appears to assume that a constitutively

active receptor cannot be further stimulated by an agonist. This, however, is not the case. Enclosed herewith are reference articles indicating that a constitutively active receptor, be it endogenous (Barker et al., Fig. 1, page 11688) or non-endogenous (Ren et al., Fig. 2, page 16485; page 16484, right column.) may be used to identify a compound as an agonist. Thus, even while in the active state, the receptor can be used to identify an agonist. The constitutively active state also enables easier detection of inverse agonist activity. Thus, use of the constitutively active version of the GPCR is valuable and useful in identifying both agonists and inverse agonists.

Applicants respectfully assert that the claims satisfy all requirements of 35 U.S.C. § 112. Withdrawal of the rejection based thereon is respectfully requested.

Claim Objections – Minor Informalities

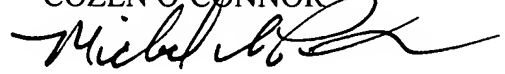
Claims 7 and 8 were objected to for containing "SEQ.ID.NO" rather than "SEQ ID NO". The claims have been corrected by amendment.

Applicants respectfully assert that all outstanding issues have been addressed and that the claims are now in condition for allowance, in light of the amendments and reasoning presented herein.

The Commissioner is hereby authorized to charge any fee or underpayment thereof or credit any overpayment to deposit account no. 50-1275.

Early reconsideration and allowance of all pending claims is respectfully requested. The examiner is requested to contact the undersigned attorney if an interview, telephonic or personal, would facilitate allowance of the claims.

Respectfully submitted,
COZEN O'CONNOR



Date: September 27, 2006

by: Michael A. Patané
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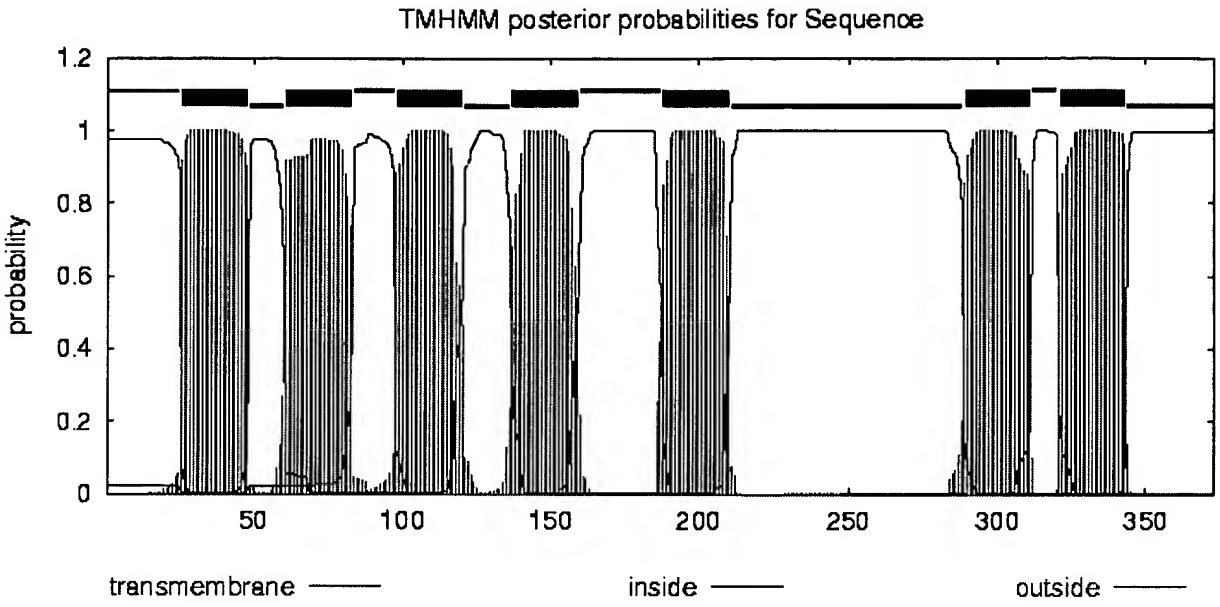
ANNEX 1



A. TMHMM result

HELP with output formats

```
# Sequence Length: 373
# Sequence Number of predicted TMHs:  7
# Sequence Exp number of AAs in TMHs: 156.69396
# Sequence Exp number, first 60 AAs:  23.36212
# Sequence Total prob of N-in:        0.02414
# Sequence POSSIBLE N-term signal sequence
Sequence        TMHMM2.0        outside      1    25
Sequence        TMHMM2.0        TMhelix     26    48
Sequence        TMHMM2.0        inside      49    60
Sequence        TMHMM2.0        TMhelix     61    83
Sequence        TMHMM2.0        outside     84    97
Sequence        TMHMM2.0        TMhelix     98   120
Sequence        TMHMM2.0        inside     121   136
Sequence        TMHMM2.0        TMhelix    137   159
Sequence        TMHMM2.0        outside    160   187
Sequence        TMHMM2.0        TMhelix    188   210
Sequence        TMHMM2.0        inside     211   288
Sequence        TMHMM2.0        TMhelix    289   311
Sequence        TMHMM2.0        outside    312   320
Sequence        TMHMM2.0        TMhelix    321   343
Sequence        TMHMM2.0        inside     344   373
```

plot in postscript, script for making the plot in gnuplot, data for plot

ANNEX 2

Conserved Tryptophan and Proline in TM6

GPCR	TM6 as predicted by TMHMM
hARE-2	YAITLLFLLL**W**S**P**YIVACYWRVF
ghrelin receptor	MLAVVVFAFILC**W**L**P**FHVGRYLF
CXCR3 chemokine receptor	LVVVVVVAFALC**W**T**P**YHLVV
5-hydroxytryptamine (serotonin) receptor 2A	LGIVFFLFVVM**W**C**P**FFITNIMAV
5-hydroxytryptamine (serotonin) receptor 2B	GIVFFLFLLM**W**C**P**FFITNITLVL
5-hydroxytryptamine (serotonin) receptor 2C	VLGIVFFVFLIM**W**C**P**FFITNILS
dopamine receptor D3	VAIVLGAFIVC**W**L**P**FFLTHVLNT
dopamine receptor D1	TLSVIMGVFVCC**W**L**P**FFILNCIL
histamine receptor H3	AVIVSIFGLC**W**A**P**YTLLMIIRAA
galanin receptor 1	TVLVVVVVFGIS**W**L**P**HHIIHLWA
neuropeptide Y receptor Y1	IMLLSIVVAFAVC**W**L**P**LTIFNTV
neurotensin receptor 1	VLRAVVIAFVVC**W**L**P**YHVRRLMF
melanocortin 4 receptor	ITLTILIGVFVVC**W**A**P**FFLHLIF
adenosine A1 receptor	LALILFLFALS**W**L**P**LHILNCITL
cannabinoid receptor 1	LVLILVVLIIC**W**G**P**LLAIMVYDV